LETTERS

Prevalence of colonisation with vancomycin-resistant enterococci (VRE) among haemodialysis outpatients in Victoria: implications for screening

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To the Editor: Patients with end-stage renal failure are a key risk group for colonisation and infection with vancomycin-resistant enterococcus (VRE). Consequently, many renal units in Australia screen these patients regularly for VRE colonisation, to assist with infection control and treatment.[1-3] Screening protocols are usually applied equally to inpatients and outpatients, even though the risk of VRE colonisation among outpatients (and therefore the cost–benefit of such screening) has not been clearly defined.

To assess the prevalence of faecal VRE colonisation among haemodialysis outpatients, we conducted a cross-sectional survey of outpatients attending 12 Victorian in-centre haemodialysis units — Austin Health (four units), Southern Health (three units) and five regional haemodialysis units (Bendigo, West Gippsland, La Trobe Valley, Central Gippsland and Bairnsdale). Patients attending these units represent about a third of the state's in-centre haemodialysis population. The study was approved by the ethics committee at each hospital, and written informed consent was obtained from all participants. All patients who attended the units between 1 October 2001 and 3 April 2002 were invited to participate.

VRE faecal carriage was assessed by three rectal swabs and one faecal specimen taken on at least three separate occasions. Specimens were inoculated onto Enterococcosel agar (BBL, Sparks, USA) containing 6 μg/mL vancomycin. All cultures were processed by standard methods for VRE identification, as described previously.[2,3]

Of 345 available haemodialysis patients, 269 (78%) consented to participate in the study (205 [76%] metropolitan, and 64 [86%] regional; participation rate per centre, 70%–91%). The 269 patients represented approximately 30% of Victorian in-centre haemodialysis patients. Overall, 74% of participants had assessment of all three rectal swabs and a faecal specimen. VRE faecal colonisation was found in three of the 269 participants (1.1%) — two were from separate metropolitan hospitals, and one from a regional centre. All isolates were *Enterococcus faecium vanB* (the most common type of VRE in Australia).[3] None of these three patients were known to be previously colonised.

This 1.1% prevalence was substantially lower than the 3.0%–4.6% prevalence previously described in renal inpatients in Melbourne,[2,3] and the 10% rate reported in the United States (where 33% of dialysis centres have one or more VRE-positive patients).[1,4] Statistical comparisons of this study with our previous two Australian studies[2,3] should be undertaken cautiously, as screening methods differed in specimen frequency, type and number. Bearing in mind this caveat, the rate of faecal VRE carriage was significantly lower among the haemodialysis outpatients in our current study compared with renal inpatients in a 1997 study by Grayson et al[2] (3/269 v 9/194; $P = 0.02$ by χ^2 test), but less definitely so when compared with renal inpatients in the 1998–1999 study of Padiglione et al[3] (3/269 v 22/739; $P = 0.09$, by χ^2 test). Since the outpatient study, screening surveys at our hospital have shown intermittent high rates of colonisation in renal inpatients and environmental contamination (unpublished data).

Given our findings in outpatients, we believe future VRE screening protocols in Australian hospitals should focus primarily on inpatients, rather than faecally continent outpatients, who have both a low rate of colonisation and low potential risk for VRE transmission or acquisition. Good compliance with practical infection control guidelines remains important to avoid widespread dissemination of VRE in our haemodialysis centres.[5]

Acknowledgements: We are grateful to the many staff and patients at the participating haemodialysis centres for their involvement, and to Kristianna Maher (Austin Hospital, Melbourne, VIC) for microbiology assistance. The study was funded by the Department of Human Services, Victoria.

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Effectiveness and side effects of thiazolidinediones for type 2 diabetes

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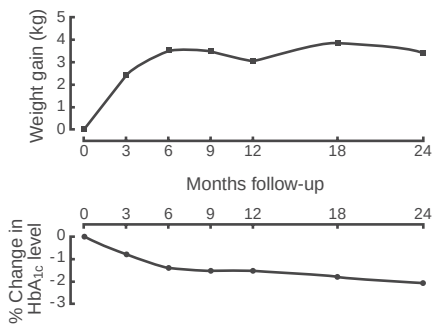
To the Editor: We read with interest the article by Hussein and colleagues on their experience with thiazolidinediones (TZDs).[1] These agents are only approved by the Pharmaceutical Benefits Scheme as part of dual therapy. We wish to present our experience of adding TZDs to metformin and sulfonylureas — hence, triple therapy — in patients with suboptimally controlled type 2 diabetes mellitus.

1 Characteristics of our 28 patients at baseline

Variable	Mean (range)
Age (years)	57.4 (31–74)
Weight (kg)	96.3 (56–137)
Body mass index (kg/m$^{2)}$	34.6 (24–50.3)
Duration of diabetes (years)	11 years (1–48)
Glycohaemoglobin (HbA_{1c}) level (%)	9.0 (7.1–10.4)

2 Changes in glycohaemoglobin (HbA_{1c}) level and weight compared with baseline values



3 Studies of thiazolidinediones added to maximal dose metformin and sulfonylurea

Variable	Roy et al[2]	Aljabri et al[3]	Dailey et al[4]	Kiayias et al[5]	Kiayias et al[5]	Byrne et al[6]	Yale et al[7]
Thiazolidinedione	Rosiglitazone	Pioglitazone	Rosiglitazone	Rosiglitazone*	Rosiglitazone†	Rosiglitazone	Troglitazone
Duration (weeks)	16	16	24	20	20	nr	24
No. of patients	48	30	181	19	19	24	101
Baseline body mass index (kg/m^2)	nr	26	32	31	31	nr	30.1
Baseline HbA_{1c} level (%)	9.3	9.7	8.1	8.9	9	9.6	9.6
Fall in HbA_{1c} level (%)	1.8	1.9	0.9	1.1	1.4	1.2	1.3
Weight gain (kg)	nr	2.6	3	4.2	4.6	0.7	0.9
% Patients withdrawn	4.2	0	5.5	0	0	0	2
% Patients with satisfactory control (HbA_{1c} level)	65 (< 7.5)	23 (< 7)	42 (< 7)	nr	nr	nr	43 (< 8)

HbA_{1c} = glycohaemoglobin. nr = not reported. * 4 mg/day; † 8 mg/day.

The records of 28 patients (15 men, 13 women) with type 2 diabetes, for whom pioglitazone was added to maximal doses of metformin and sulfonylurea because of suboptimal control, were reviewed. Baseline patient characteristics are shown in Box 1. Mean follow-up was 9.6 months (range, 3–24 months); the average pioglitazone dose was 31.3 mg. The mean fall in the level of glycohaemoglobin (HbA_{1c}) was 1.26% — 10 patients achieving an HbA_{1c} level of less than 7% at last review. Four patients did not respond to therapy; none withdrew because of side effects. Eight patients whose HbA_{1c} fell less than 0.5% after 3 months continued taking pioglitazone, achieving an average fall in HbA_{1c} of 1.25% after a mean of 12 months follow-up. Mean weight gain was 3.35 kg (–3.2 kg to 11.6 kg). Mean changes in HbA_{1c} level and weight compared with baseline over 24 months are shown in Box 2. There was no correlation between these outcomes, and no baseline characteristic predicted glycaemic response.

Six studies have reported the efficacy of TZDs in triple therapy (Box 3), and show a consistent fall in HbA_{1c} level at the expense of weight gain, with a low rate of withdrawals because of adverse effects. Our findings were similar to those of these previous reports in terms of glycaemic response and low rate of side effects. The much higher rate of side effects reported by Hussein et al[1] is likely to be the result of the coprescription of TZDs with insulin in 64% of patients in their study. While fluid retention has been reported in up to 5% of patients taking TZDs as monotherapy or in combination with oral hypoglycaemics, 15% of patients using TZDs with insulin may develop significant oedema. Most reports describing precipitation of cardiac failure with TZDs have been in patients using combination therapy with insulin. It would be interesting to know what proportion of the patients who developed peripheral and pulmonary oedema in the study by Hussein et al[1] were also receiving insulin. One prospective randomised trial comparing the addition of pioglitazone and bedtime insulin to maximal metformin and sulfonylurea found similar efficacy in improving glucose control, but less hypoglycaemia and improved high density lipoprotein cholesterol levels with pioglitazone.[3] A study of the long-term efficacy of triple therapy found 26 of 35 patients (74%) had good control after a mean follow-up of 37 months, their HbA_{1c} level having fallen from 8.7% to 6.9%.[8]

In conclusion, the experience of our unit and the published literature is that TZDs are efficacious in improving suboptimal diabetic control in patients on maximal doses of metformin and sulfonylurea. Eight individuals in our group had a significant improvement in control subsequent to minimal response after the initial 3 months of treatment, suggesting a longer trial of TZDs should be employed before classifying patients as non-responders. It is to be hoped that the regulatory authorities will allow the use of TZDs in triple therapy.

Competing interests: Both authors have received honorariums from Eli Lilly for presentations.

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To the Editor: It was with interest that we read the recent article on real-life experience with thiazolidinediones by Hussein et al.[1] The authors noted the absence of severe liver toxicity with the newer agents, rosiglitazone and pioglitazone, in contrast to troglitazone, which was withdrawn because of cases of hepatic failure.[2] However, the sample was too small to conclude that these thiazolidinediones (TZDs) carry no hepatic risk, and they endorsed the current Pharmaceutical Benefits Scheme recommendations that liver function tests (LFTs) be monitored every 2 months. We have collected similar clinic data showing that the development of significant abnormalities in results of LFTs with TZD therapy is uncommon, and in fact, there are often improvements in LFT findings.

We reviewed the files of 166 patients with type 2 diabetes treated with TZDs between 1 August 2000 and 30 November 2002, with the aim of assessing their long-term effect on LFT results. Therapy was discontinued within 3 months in 26 patients. The reasons were non-compliance (7), therapy ineffective (8), weight gain (5), dyspnoea (1), peripheral oedema (1), malaise (1), dizziness (1), angio-oedema (1), and pre-existing LFT abnormality (1). We analysed data on the remaining 140 patients (see Box) treated for a mean of 188±4 days with either pioglitazone (109 patients) or rosiglitazone (31 patients).

All LFT results improved significantly (Box). At baseline, 90 patients had abnormal findings on LFTs. These findings normalised in 43 of these patients (including one with steatohepatitis proven on biopsy); improved in 29 patients; and were unchanged in nine patients. LFT findings deteriorated in nine patients, leading to cessation of therapy in two. Most patients with normal LFT results at baseline experienced improvements of these parameters within the normal range. Three patients developed new abnormalities in their LFT findings, and therapy was stopped in one patient, leading to resolution of LFT abnormalitites. Changes in glycohaemoglobin (HbA_{1c}) levels correlated positively with changes in activity of alkaline phosphatase (correlation coefficient [r], 0.33; $P < 0.01$), aspartate aminotransferase (r, 0.27; $P < 0.01$) and alanine aminotransferase (r, 0.29; $P < 0.01$).

Our findings support those of Hussein et al, that TZD therapy is usually stopped for reasons other than hepatic dysfunction. In contradistinction to early concerns about their hepatic safety, the improvements in LFT findings seen in our patients suggest that TZDs may even benefit hepatic function. In patients with diabetes, abnormal findings on LFTs are often attributed to fatty liver, which predisposes to steatohepatitis. TZDs may well alleviate or prevent steatohepatitis,[3] thereby providing benefits beyond that of improved glycaemic control, and mild abnormalities in LFTs should not discourage their use in patients with diabetes.

Acknowlegements: We acknowledge the contribution of Dr Rob Coles, who supplied much of the data for our study.

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Changes in liver function and glycohaemoglobin (HbA_{1c}) level

Variable	Baseline	6-month follow-up	Change	*P**
Weight (kg)	93±2	96±2	3±0.4	< 0.001
HbA_{1c} (%)	8.9±0.1	7.9±0.1	−1.0±0.1	< 0.001
Albumin (g/L)	41±0.2	41±0.2	−0.5±0.2	< 0.02
Bilirubin (μmol/L)	9.4±0.4	8.5±0.3	−0.9±0.3	< 0.003
ALP (U/L)	92±2	79±2	−13±2	< 0.001
GGT (U/L)	44±3	31±2	−13±2	< 0.001
AST (U/L)	26±1	22±1	−3±1	< 0.001
ALT (U/L)	33±2	25±1	−8±2	< 0.001

ALP = alkaline phosphatase. GGT = γ-glutamyl transferase. AST = aspartate aminotransferase. ALT = alanine aminotransferase. * Paired *t* test.

Profound hypocalcaemia after zoledronic acid treatment

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To the Editor: Zoledronic acid is a new bisphosphonate treatment for hypercalcaemia of malignancy, multiple myeloma and documented bone metastases from solid tumours, in conjunction with standard chemotherapy.[1] Transient hypocalcaemia, a side effect of bisphosphonate therapy, can occur with zoledronic acid.[2]

We describe a patient with transient severe hypocalcaemia after zoledronic acid treatment.

An 88-year-old white woman with multiple myeloma presented with left hip pain. Imaging revealed multiple lytic lesions in the spine, pelvis and upper femurs. She was commenced on zoledronic acid 4 mg and a 5-day course of melphalan 10 mg/day and prednisolone 100 mg/day. At this stage, her serum calcium concentration was 2.38 mmol/L (normal, 2.15–2.55 mmol/L) and serum phosphate concentration was 0.8 mmol/L (normal, 0.8–1.5 mmol/L). She had renal impairment with reduced creatinine clearance of 35 mL/min.

Nine days later, she underwent surgical repair of a duodenal ulcer. Post-operatively, she was found to be hypocalcaemic, with serum calcium concentration of 1.34 mmol/L and ionised calcium concentration of 0.78 mmol/L (normal, 1.14–1.29 mmol/L). Serum phosphate concentration was 0.4 mmol/L and magnesium concentration was 0.87 mmol/L (normal, 0.70–0.90 mmol/L). Her serum 25-hydroxyvitamin D concentration was 14 nmol/L (normal > 50 nmol/L) and her parathyroid hormone level was 44 pmol/L (normal, 1.5–8.0 pmol/L). The patient displayed no symptoms of hypocalcaemia and had negative Chvostek and Trousseau signs. Her QTc interval was normal. Her hypocalcaemia was managed with oral calcium carbonate 4.5 g/day and calcitriol 0.5 μg/day. Ten days later, her total serum calcium concentration rose to 2.31 mmol/L and her phosphate concentration was 0.9 mmol/L.

Bisphosphonates inhibit osteoclast-mediated bone resorption, thereby reducing serum calcium concentration. Compensatory secondary hyperparathyroidism prevents significant hypocalcaemia by

enhancing renal calcium conservation, 1,25-hydroxyvitamin D production, and osteoclastic bone resorption.[1,2] In our patient, the 1,25-hydroxyvitamin D concentration was not measured. However, in view of the low 25-hydroxyvitamin D and impaired renal function, this level may have been low, further exacerbating the hypocalcaemia.

Transiently low phosphate concentration in this patient may be due to fasting and co-administration of 5% dextrose, as our patient was fasted for 3 days peri-operatively and given a combination of intravenous 5% dextrose and normal saline.

Vitamin D insufficiency afflicts a large proportion of the elderly population,[3] and its existence needs to be recognised before commencement of bisphosphonate therapy, so that adequate calcium and vitamin D supplementation can be given to reduce the occurrence of hypocalcaemia.

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Impact of smoking, diabetes and hypertension on survival in the elderly: the Dubbo Study

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To the Editor: The informative study by Simons and colleagues[1] has highlighted a major concern. Chronic obstructive pulmonary disease (COPD) is one of Australia's top four causes of death and burden of illness,[2] yet the authors have made no mention of COPD. Failure to recognise the importance of this disease is an endemic attitude in Australia and globally[3-5] that results in under-representation of COPD in epidemiological surveys and in inadequate funding for effective treatments and research.

The study found that peak expiratory flow (PEF) provides the highest hazard ratios for predicting time to death in women (and the second highest in men). There is even a "dose–response" effect. Using the term "impaired PEF" is a bit like saying "impaired ECG" without attributing a diagnosis. PEF is a measure of airway calibre, and impairment of PEF indicates airway disease — largely COPD in this population. Smoking accounts for about 85% of the risk of COPD, and about 50% of smokers develop airflow limitation,[4-6] so it is not surprising that the interaction between PEF and smoking was the most important predictor of reduced survival in this large cohort.

It's time to stop hiding our heads in the ashtray! Smoking combined with low PEF *is* COPD. We must demand that our medical and epidemiological professions uncover people with undiagnosed COPD. Early diagnosis is simple.[5] It's not normal to be unable to keep up with friends at work or during recreation because of breathlessness, and a daily cough is really an airway disease. If symptoms are acknowledged, spirometry will confirm the diagnosis. We should help our patients to enunciate these hidden symptoms so their condition can be diagnosed accurately, and effective management begun, as highlighted in the "COPDX management guidelines".[5] Primary and secondary prevention must focus on reducing smoking among young people. Smoking cessation, the use of effective drugs, and pulmonary rehabilitation are the cornerstones of COPD therapy that lead to better quality survival.

COPD is common and under-diagnosed. Simons et al have partly exposed this deadly condition. Their data, added to other Australian data,[7] should trigger actions that facilitate earlier diagnosis throughout Australia and support delivery of effective treatment to the thousands "dying a slow death" from COPD.

Australia's illness burden from COPD is high. Its prevalence and burden in Australia are rising, especially in women. Globally, the World Health Organization expects COPD to rise from 12th to 5th as a cause of illness burden by 2020.

We must acknowledge that PEF impairment is not simply a mysterious risk factor for early "all-cause mortality", but is indicative of COPD being a major contributor to death in this population.

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In reply: The prospective Dubbo Study of the elderly has produced a series of publications in which reduced peak expiratory flow (PEF) has been shown to be associated with increased risk of death,[1,2] as well as increased risk of heart attack,[3] ischaemic stroke[4] and admission to a nursing home.[5] We have employed a purely statistical definition of impaired PEF, namely the lowest third of our sex-specific population distribution. We agree that many subjects so defined with impaired PEF, and who are smokers, will have underlying and potentially undiagnosed chronic obstructive pulmonary disease (COPD).

Epidemiological studies have highlighted the importance of impaired PEF. It is now time for health professionals to implement the COPDX management guidelines referred to by Frith[6] in a still more effective manner and to devise better prevention programs.

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Working with registrars: a registrar's perspective

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To the Editor: Up and coming surgical registrar, Ken Wong, presents a revolutionary plan to allow him to look after surgical patients in the operating theatre.[1] The use of the telephone for communication has merit, but he won't feel so smug when he gets to the chapter entitled "The management of surgical patients in NSW public hospitals in winter". There will be nowhere for Dr Wong to "hide" when he realises our operating theatres are, in fact, solar powered and that, when the sun goes down in winter, the theatres conk out. Surely now, with the statewide mergers of health services, there will be enough excess "committee people" to form a collaboration with the western NSW farmers so that the mice plague can be harnessed and trained to run on the cogs and at least provide lighting during power shortages. I'm sure my daughter could lend a few cats to chase the mice. In the absence of the provision of more hospital beds, the substitution of cat-and-mouse power for solar power is the best "winter strategy" I've heard in the last 10 years. This concept will feature in our next chapter, "How to train surgeons without patients or operating time".

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The Medical Journal of Australia (MJA) is published on the 1st and 3rd Monday of each month by the Australasian Medical Publishing Company Proprietary Limited, Level 2, 26-32 Pyrmont Bridge Rd, Pyrmont, NSW 2009. ABN 20 000 005 854. Telephone: (02) 9562 6666. Fax: (02) 9562 6699. E-mail: medjaust@ampco.com.au. The Journal is printed by Offset Alpine Printing Ltd, 42 Boorea St, Lidcombe, NSW 2141.

MJA on the Internet: http://www.mja.com.au/

Published in 2 volumes per year.
Annual Subscription Rates for 2005 (**Payable in Advance**) to: **AMPCo**, Locked Bag 3030, Strawberry Hills, NSW 2012
Individual Subscriptions (includes 10% GST)
Australia: $A319.00, Medical students (Australia only): $A60.00
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Indexes are published every 6 months and are available on request as part of the current subscription.
Single or back issues contact: **AMPCo (02) 9562 6666.**

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28,516 circulation as at 30 September, 2004

ISSN 0025-729X